ATONIC (RELAXED) PARAPLEGIA CAUSED BY COMPRESSION OF THE SPINAL CORD.

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Concluded.

AUTOPSY.

Microscopic Examination.—Upon opening the spinal canal the dura-mater was found to be very thick at the level of the gibbosity extending over a length of 4 to 5 centimetres, and adherent to the posterior surface of the vertebral bodies. It was affected by a caseous degeneration visible upon the external surface ; its internal surface, on the contrary was smooth and did not adhere to the pia-mater. The spinal cord compressed to the level of the centre of this pachymeningitis seemed a little shrunken, but presented no manifest alteration exteriorly, nor upon a transverse cut. Above and below the affected region in the pyramidal tracts and in the columns of Goll, in a fresh condition, nothing abnormal could be seen. After hardening in Müller's fluid, on the transverse section, the area of the pyramidal tracts more particularly to the right and below the compressed region, could be distinguished from the neighboring parts by a lighter color; there seemed to be a slight alteration there.

The vertebral substance of the 4th and 5th dorsal vertebræ adhered to the dura-mater and was friable.

In the upper part of the lungs tubercular granulations were found. There were no abnormal indications in the heart, liver or spleen.

The bladder and ureters, contained urine mixed with pus. The left kidney was transformed into a cyst filled with a seropurulent liquid and the calices were distended ; the renal parenchyma was notably atrophied. The right kidney was of normal size and presented traces of amyloid degeneration.

The left femoral vein was filled by a clot which adhered slightly to its wall.

Microscopic Examination.—In the portions of the spinal cord at the level of the centre of the pachymeningitis, and below in different portions of the pyramidal tract and in the columns of Goll, granulated bodies were very rare and seemed no more numerous than in the normal state. After hardening the spinal cord in Müller's fluid, transverse sections were made which were colored some by Weigert's process and others with carmine. At the level of the centre, the interstitial tissue in the area of the lateral tracts seemed more abundant than in the normal state; the nerve fibres on the contrary, were more scattered, but the lesions were slightly marked. The columns of Goll were normal above the compressed region. Below this region the pyramidal tracts were a little sclerotic. This sclerosis, the reality of which, on the left side, may be doubtful, was incontestable on the right; the lesion was however, very slight, much less marked, for example, than that which develops habitually after hemorrhages of the internal capsule. All the other parts of the spinal cord were absolutely normal.

Here, then, is a man suffering during a month and a half with a complete motor paraplegia with amyotrophy and without anæsthesia, with vesico-rectal troubles and eschar of the sacrum; the paraplegia was atonic and the tendon reflexes were completely abolished. Microscopic examination has shown that the spinal cord in the compressed region, presented only slightly marked lesions and that the consecutive sclerosis of the lateral tracts was quite rudimentary. Death should be attributed to poisoning caused by amyloid degeneration of the kidney and the infection provoked by the pyelo-nephritis. But, from the particular point of view which occupies us, this is of no special interest.

CASE II.—Louis Moreau, aged 71 years, a shoemaker, admitted August 8, 1870, in the ward Rayer. There was nothing interesting to report either in his hereditary antecedents or in his personal history farther back.

The first morbid manifestation which seemed to have any relation to the present affection appeared fifteen months ago. One day as he was walking in the street, he felt a giddiness and fell to the earth without entirely losing consciousness; passers-by assisted him and he was able to walk home; the next day he felt very much depressed, but the day after resumed his work.

From that time he had frequent attacks of giddiness which accompanied vertigo and made it necessary for him to catch at some support in order to avert a fall; he once fell into a cellar receiving a wound of which he still carries the scar.

April 5th, 1890, he fell, as the result of giddiness, in the Rue St. Honoré and could not rise; he was taken home in a carriage; although he was not then in a condition to walk, he noticed that he could move his limbs about in bed. The next day he observed that the right side of the body (both upper and lower limb) was weaker than the left, and perhaps a little less sensitive; but there was no change in the muscles of the mouth nor embarrassment in speech. He was taken upon a stretcher to the central office and was admitted to the Hôtel-Dieu annex, under the care of M. Chartreusse. About ten days after the last fall his situation began to ameliorate a little; he was able to rise and take a few steps in the hall; but this improvement lasted but a few days; he was soon obliged to keep his bed and never left it again.

From this time his condition was about stationary in all that concerns, at least, the paralysis of the lower extremities. The paresis of the right arm, on the contrary, after having persisted during several weeks diminished and finally disappeared.

This following is the report from the time of the admission of the patient to La Pitié:

It was impossible for him to rise and stand erect; at the same time the paraplegia was not complete. He could with great effort bend the thighs slightly, and the legs, but he was absolutely incapable of lifting the limbs above the level of the bed. He could not raise his feet; it was only with great difficulty and feebly that he could make a movement of flexion. The lower limbs were completely relaxed; the muscles were shrunken, but there was no localized amyotrophy. The electro-faradic contractility appeared normal. The tendon reflexes were very weak especially on the right, the foot clonus was lacking. Sensibility was preserved in all its forms.

The upper extremities were weak, but all the movements were capable of being executed in the normal manner. The thenar eminence was shrunken on both sides. There was no facial paralysis, and no difficulties of speech. In the sacral region there was diffuse redness and a superficial eschar of the dimensions of a five franc piece.

The patient insisted that he felt the calls of nature but was obliged to satisfy them immediately. The functions of the sphincters of the bladder and anus were weakened ; it was not a case of incontinence from retention for the bladder was not distended and there was no retention of urine. There was no distortion of the vertebral column and it was not painful to the touch or to pressure. No renal troubles ; nor *arcus senilis*. The intelligence seemed unimpaired, but the memory was enfeebled. The arteries were atheromatous. Examination of the other viscera, thoracic or abdominal, revealed nothing abnormal. The urine contained neither sugar nor albumen. The patient has been examined at intervals ever since his admission, and it is reported that the paraplegia becomes more pronounced while preserving the characteristics noted in the beginning : the weakening of the tendon reflexes is maintained and the lower limbs preserve their flaccidity ; at the same time it is impossible to extend the left leg completely upon the thigh ; the muscles are in a state of complete relaxation, but when the completion of the extension is desired and a certain limit is passed, there is a sudden resistance which is manifestly due to fibro-tendinous retractions.

Sept. 8th.—The patient complained of difficulty in deglutition and respiration. The examination of the lungs gave a negative result. These troubles persisted during the following days.

Sept. 12th.—In the morning the patient was found in a coma ; the respiration was accelerated ; the rectal temperature was 40.9° C. This condition continued for twenty-four hours, and September 13th the patient died.

AUTOPSY.

Microscopic Examination.—The cerebral meninges and the cerebrum examined upon the surface and upon many transverse sections presented nothing abnormal, except that the arteries of the base are atheromatous.

The spinal dura-mater in the cervical region, at the level of the 4th, 5th, 6th and 7th pairs adhere strongly to the posterior face of the vertebral body and it was very much thickened ; when a longitudinal section was made of it, it was seen to be doubled in two leaves between which was found a layer of yellowish white pus, very dense. The spinal cord presented a depression in this region ; it was injected at its surface, but it was detached as in the normal state from the dura-mater which was smooth on its inside face. The other parts of the spinal cord above and below the centre of compression, at their surface and upon transverse sections, bore a normal aspect both in the fresh state and when the spinal cord was hardened in Müller's fluid.

The vertebral column was not distorted. The vertebræ examined with care in the region where the purulent centre existed, seen upon their external and internal surfaces and upon their sections to be free from alterations. The heart was surcharged with fat ; the left ventricle was hypertrophied ; no valvular lesions ; the aorta was atheromatous. The lungs were congested at the bases and were emphysematous. The liver was slightly sclerotic. Nothing to report as to the spleen or the gastro-intestinal canal.

Microscopic Examination.—An examination was made of the portions of the spinal cord in a fresh state at the level of the lesion ; below the centre, in the different parts of pyramidal tracts, and above the centre in the tracts of Goll.

The tissue did not seem to be altered ; no granulous bodies were found, or at least, if they were discovered in certain preparations, they appeared no more numerous than in the normal condition.

After hardening the spinal cord in Müller's fluid, transverse sections were made which were colored, some by Weigert's process and others with carmine. Careful examination of these different sections, whether from the pachymeningitis or from parts of the spinal cord situated above the centre or below it, in the dorsal region or in the lumbar region did not show a well defined lesion. It might be said that at the level of the centre of compression the neuroglia was a little thicker than usual, and that the same was true of the neuroglia of the pyramidal tract in the dorsal region ; but, if these alterations did exist, which is doubtful, they were very small, especially if the age of the subject is taken into account.

This examination may be summed up as follows : The patient was subject for more than a year to attacks of vertigo accompanied by weakness of the lower limbs; these symptoms were at first transitory.

But one day after an attack of giddiness, the patient fell and was unable to rise ; he was attacked at the same time with paraplegia and right hemiplegia with face unaffected. The weakness of the right arm disappeared immediately, but the lower limbs were paralyzed, seriously though not completely; the patient was able to move very little. There were at the same time some vesico-rectal troubles and a superficial eschar upon the sacrum. The legs preserved their sensibility and the muscles were not atrophied. They were relaxed and the tendon reflexes were enfeebled. This paraplegia persisted, without modification, during five months or until the death of the patient.

The microscopic examination established the anatomical integrity of the spinal cord. The fever which developed toward the last seems to indicate that the patient succumbed to infectious disease; but the histological and the bacteriological study of the viscera not having been made it is impossible for us to be certain in this respect. This side of the case is of no interest from the point of view which occupies us.

The two following cases, for one of which I am indebted to Kadner[1] and for the other to Druschky, although very succinct, merit a place with the two preceding cases.

CASE III.—From Kadner's Memoir. Carcinoma of the upper four dorsal vertebræ. Relaxed paraplegia with disturbance of sensibility, retention of urine and eschar. The motor paraplegia was complete during seventeen days and during that time the reflex of the Achilles tendon was weak and the patellar reflex was lacking. The autopsy showed that there was no secondary degeneration.

CASE IV.—Druschky's. In a case of compression of the spinal cord following an external meningitis located between the second and sixth dorsal vertebræ, the foot clonus was absent. The autopsy showed that there was no secondary degeneration.

The author enters into no further details on this subject. The considerations into which I entered in the first part of this work permit me now to be brief. The consequences which follow from the observations just related seem to me manifest.

We have long known that a paraplegia of spinal origin may be due to a simple compression of the spinal cord, without destruction of the tissue ; the well known examples of paraplegia resulting from vertebral fractures or luxations, disappearing after their reduction, furnish proof of it ; but in this case the paralysis is of short duration. But it is scarcely admitted that a paralysis of long standing could result in the same manner, or at least, this idea is not yet classic. Some observations show however, that such might be the case. M. Joffroy[2] observed a case of compression of the lumbar enlargement and of the equinus by a secondary sarcoma of the *dura mater* having given rise, during more than four months to a complete paraplegia, without any lesion of the nerves nor of the spinal cord.

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[1] Kadner. Zur Casuistik des Ruckenmarks-compression. Arch. d. Heilk. xvii. 1876. s. 489.

The facts which we relate establish clearly that the compression of the spinal cord in the dorsal or cervical region, may, without determining an appreciable organic lesion produce a very marked and even complete motor-paraplegia of several months duration, which is accompanied by vesico-rectal troubles and trophic alterations of the skin. The tracts of the spinal cord may then, under the influence of compression, lose their physiological properties, without being modified in any apparent manner in their structure. This remark does not apply, it is true, except to centrifugal fibres and more especially to motor fibres ; it is not the same with sensitive fibres, for in one case anæsthesia was completely wanting, and in this case the sensibility of the paralyzed members was almost completely preserved.

The pathology of the peripheral nerves furnishes us with facts which may be placed with the preceding ; the radial motor paralysis resulting from a compression may last several months without being accompanied with troubles of sensibility and without, in all probability, any notable alteration of the nerve fibres.

On the other hand these facts show that a paraplegia resulting from a spinal compression may remain relaxed during several months without any alteration of the gray matter of the lumbar spinal cord, without the alteration of the external radicular zones at this level, without lesions of the peripheral nerves and without the destruction of a portion of the spinal cord in its whole diameter.

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[2] JOFFROY. *Bulletins de la Société Anatomique*, 1871.

It seems to me impossible to interpret these facts and to determine the cause of this flaccidity.

The anatomical examination showed, in effect, that the pyramidal tracts were in a state of complete integrity or nearly so. Now, we have seen that in the immense majority of cases of compression of the spinal cord the spasmodic phenomena have for an anatomical substratum lesions of the pyramidal system. It is not surprising, therefore, that the exaggeration of the tendon reflexes is lacking.

But we must question as to why the inverse phenomenon developed, and why the tendon reflexes were weakened. We think it will be permissible to consider this due to the cachectic condition of the patients.

I call attention to the fact that it would be illegitimate to conclude from our observations that a spasmodic paraplegia due to a spinal compression is necessarily connected with organic lesions of the pyramidal system. Such an affirmation would be contradicted by Colman's case which has been cited ; in this case it is a question of paraplegia by compression with exaggeration of the tendon reflexes and epileptoidal tremors ; the anatomical examination has shown that the spinal cord was normal.

The comparison of these dissimilar facts gives cause for reflection. It is not illogical to sustain as a hypothesis, that according to the intensity of compression to which the nerve fibres are subjected, according to the general condition of the patients, the centres of the tendon reflexes are submitted to a dynamic action or an inhibitory action.

Whatever be the interpretation given to these phenomena their reality cannot be contested. It may be remembered in connection with this subject, that in disseminated sclerosis, an affection in which the nerve fibres are also subjected to compression, the tendon reflexes may be, as in the case which we are studying, normal, exaggerated or abolished.

Then the spasmodic condition of a paraplegia by compression does not constitute the characteristic mark of an organic lesion of the spinal cord.

We have seen on the other hand, that the flaccidity of a paraplegia does not constitute a certain indication of the integrity of the spinal cord. But we have been able to determine the circumstances in which a relaxed paraplegia can be connected with a spinal lesion. We have insisted sufficiently on this point not to return to it; and we have the right to deduce from this study the following conclusions: When a relaxed paraplegia caused by spinal compression lasts more than a month, when the arc of the tendon reflexes presents no lesion, when no portion of the spinal cord is destroyed throughout its diameter, there is reason to suppose that the nerve fibres of the spinal cord are intact, or at the most that their alterations are very slight.

I have not the right however, to formulate, at this time, such a proposition without making some reservations.

I have already announced the hypothesis that the dynamic influence exercised upon the centres of the tendon reflexes by a secondary sclerosis, may be annihilated by a cause which will act in an inverse or inhibitory direction; it is not irrational to suppose, for example, that in case of a diabetic or a cachectic attacked by descending degeneration the reflexes would not be exaggerated. There may be other unforeseen causes which will counterbalance the spasmodic action of the secondary sclerosis.

Nevertheless, the preceding conclusion may be accepted provisionally.

It seems to me that it will be henceforth possible, in taking these observations into account, to establish clinically, in analogous cases an exact diagnosis.

Let us suppose, for example, that we have to do with an atonic paraplegia resulting from an alteration of the upper or middle dorsal vertebræ. Let us admit that this paraplegia has already lasted several weeks, and that none of the signs exist which denote the alteration of one of the parts of the arc of the tendon reflexes (motor nerves, sensitive nerves, external radicular zone, gray matter and anterior cornua).

The problem is immediately defined and there remains to be determined only, whether the flaccidity is due, as in the cases of Bastian and Bowlby, to a serious lesion, or as in my observations, to the absence of organic lesions. If these two orders of facts present, from an anatomical point of view, a striking antagonism, it is none the less true that there may exist great clinical analogies between them, for in either case the motor paraplegia and the abolition of the tendon reflexes may be absolute. In any case the diagnosis would be easy, for in the facts of the first group, the paraplegia is accompanied by a profound anæsthesia, while in the observations of the second group the paralyzed members preserve their sensibility more or less completely.

Is it necessary to call attention to the fact that it will be of great practical interest to make in cases of this kind a precise diagnosis.

It can be admitted, *à priori*, that other things being equal, the prognosis will be less grave when it has been established that nervous troubles exist which are due to a superficial compression of the tissues, that they are not seriously disorganized and that secondary degenerations are wanting. It is true that the existence of more marked alterations, of sclerosis of the pyramidal tracts, does not imply a desperate prognosis, and it is not rare, especially in Pott's disease, to see persons affected with spasmodic paraplegia, recover in time the use of their limbs. One of the finest observations of this kind was made in the service of Prof. Charcot, at the Saltpetrière, and published by M. Michaud. It was the case of a patient who, after having been attacked with spasmodic paraplegia, which reduced her to absolute impotence, was cured, and was able, at least for a time, to walk without cane or crutches; she remained in this condition four years and succumbed then to the effects of coxalgia; the examination of the spinal cord revealed marked alterations, which were located above the centre, in Goll's tracts, and below the pyramidal fascicles. There will be found also, in M. Charcot's Tuesday Lessons[1] the account of an extremely interesting case in which the recovery of a spasmodic paraplegia, from Pott's disease which had lasted three years, has been permanent for twenty years, and in this long period there has been nothing abnormal observed in her walk.

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[1] Tuesday Lessons 1888, 1889, p. 176 and following.

However, if analogous observations were always collected with care, it would be perhaps seen that there would not be an absolute return to the normal condition. This is the opinion of Prof. Charcot, who expresses himself thus on this subject : " How can we believe that a spasmodic paraplegia, evidently caused by the slow compression of the spinal cord, a compression which necessarily, to the best of our knowledge, has produced in the nervous tract, at the level of the point compressed, lesions of transverse myelites; how can we believe, I say, that a paraplegia of this order, dating back three years, and having persisted in the highest degree during a period of one year, can be cured without leaving an apparent trace? This point I will return to in an instant. I haste to mention, gentlemen, that this integrity, which might be thought absolute, of the lower limbs, is but a deceptive appearance. Spasmodic paraplegia persists in some form in a rudimentary condition, in cases which seem completely cured of the spinal compression, caused by Pott's disease. This latent paraplegia, if one may call it so, may not, as was seen in the case of B——t, manifest itself by any anomaly in the walk, but is revealed by the aid of certain explorations, which bring to light significant indications. In the case of B——t, each time I have made him the subject of a demonstration, I have reported the existence in the lower limbs of a tremor when the foot was straightened, which was quite marked, and since I have learned to recognize Westphal's sign, a manifest excitement from the shock produced in the leg by the pressure of the rotulian tendons.

"Well, gentlemen, I will state, from having seen it many times clinically, that when the case is of this sort, whatever the spinal affection in question, the spasmodic paraplegia really exists in power, in a condition of opportunity," as M. Brissaud has said, "and often only an exciting cause is needed, accidental in appearance, to realize definitely permanent rigidity, under a more or less marked form."

On the contrary,in cases similar to those which we publish here, it is easy to conceive the possibility of real and complete cure. The lesions of the spinal cord being, as has been seen, nul or quite rudimentary, and the functional troubles being, so to speak, purely dynamic, it is rational to admit that if the agent of the compression disappears, or if the process of pachymeningitic inflammation has been simply checked, at a given moment the functions might be re-established in all their integrity. He resorts then to bringing these two orders of facts together, to which he is not indifferent from the point of view of prognosis, to learn to distinguish one from the other.

It may be urged that it is of importance from a therapeutic point of view, to make an exact diagnosis, and to know how to recognize the varieties of paraplegia, with which we are occupied.

I think in fact, that the resection of the spine, followed by the removal of the tissues which confine the spinal cord, would be especially suited to cases of this order. There is reason to admit that in these patients the organic resistance of the spinal cord is not unlimited, and it is permissible to suppose that in time the compression might provoke the development of spinal lesions. On the other hand, it is rational to suppose that in removing this compression, the disappearance of the functional trouble depending on it will follow.

The operation seems to me to respond to a double indication, it may be considered as a preventive means and as a curative process. It is self-evident that surgical intervention may be inadvisable from circumstances independent of the paraplegia and arising from a bad general condition.

The conclusions which it seems to me may be drawn from this study, are as follows :

1st. A compression of the spinal cord may, without producing an appreciable lesion of the organ, give rise to an intense and perhaps complete paraplegia, capable of being prolonged for several months.

2d. When, in atonic paraplegia of a duration longer than a few weeks, resulting from a compression of the spinal cord, the flaccidity can be attributed only to a lesion occupying some part of the reflex arc, and is also not due to the destruction of a portion of the spinal cord in its entire diameter, it is permitted, for the present, to believe that the spinal cord is not changed, or only slightly changed.

3d. There is reason to admit that this variety of paraplegia, aside from extrinsic circumstances, permits a relatively benign prognosis.

4th. When the general condition of the invalid is not opposed to it, surgical intervention seems particularly indicated in cases of this kind.

DISSEMINATED SCLEROSIS.

In the "Archives of Ophthalmology" for July, 1891, Zimmermann reports the following case:

W. F., painter, æt. 26, complaining of dimness of sight in right eye for eight weeks, and also of diplopia. Vision, R. = $^{15}/_{70}$, L. = 1. Field of vision and perception of colors normal. Both pupils normal. Upon *ophthalmoscopic examination the temporal two-thirds of both discs were found to be perfectly white.* There was crossed diplopia from paresis of the right internal rectus. Under iodide of potassium and hypodermic injections of strychnia, vision improved. Two months later vision was normal in both eyes, and the diplopia had disappeared. The ophthalmoscopic appearances, however, remained unchanged. Four years later the patient had developed a primary spastic paraplegia, and subsequently many of the symptoms of a disseminated sclerosis.

He states in conclusion that *atrophy of the optic nerves, when associated with the symptoms of primary spastic paraplegia, is the most valuable and reliable guide in the diagnosis of disseminated sclerosis.* W. M. L.